HISTOPATHOLOGY OF CALCIFICATION OF THE SPINATUS TENDONS AS ASSOCIATED WITH SUBACROMIAL BURSITIS.

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Under the title "Prevalent Fallacies Concerning Subacromial Bursitis; its Pathogenesis and Rational Operative Treatment," Brickner has described[1] his observations, surgical and radiographic, of calcareous deposits in and upon the supra- and infraspinatus tendons, associated with adhesive subacromial bursitis. The following is based upon the histological study of portions of diseased tendons in four of his cases.

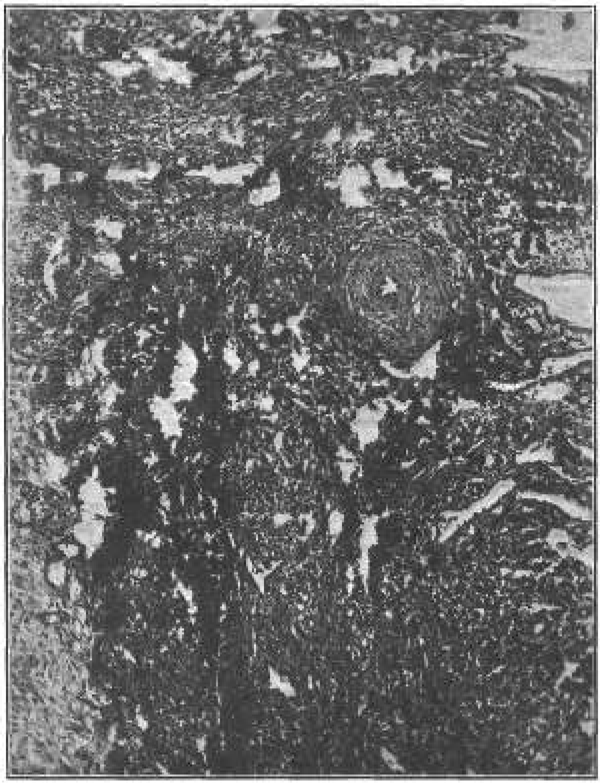

Fig. 1.—Case 1. Showing intratendinous formation of young granulation tissue and formation of new bloodvessels. To the left, a narrow zone of older organized connective tissue.

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[1] Amer. Jour. Med. Sci., March 1915, cxlix, 351.

CASE I.—Female, aged forty-five years. Operation upon the sixteenth day after indirect trauma. Fresh adhesions in and ecchymosis of bursa; yellow, semiliquid calcareous deposit overlying and within laceration of swollen supraspinatus tendon; infraspinatus infiltrated with blood.

Pathological Examination (Figs. 1 and 2). Section shows a crescentic mass of tissue. Covering the entire convex border is a narrow zone of fairly loose connective-tissue fibers—a comparatively sparse number of large, long spindle-shaped cells of the connective-tissue type, a fair sprinkling of round cells, a few red blood cells, a few thin-walled bloodvessels. This tissue represents a fairly well-organized granulation tissue.

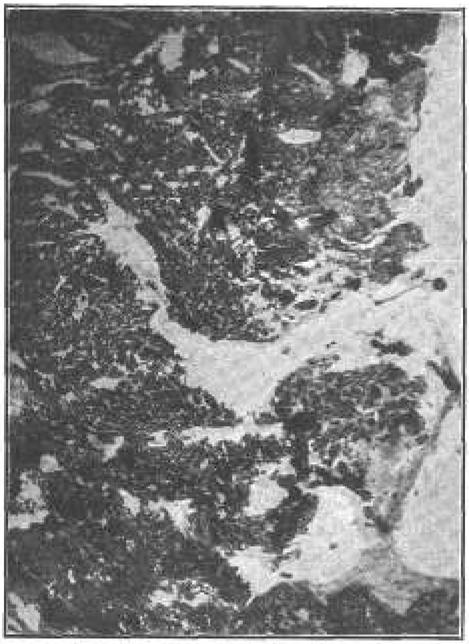

FIG. 2.—Case 1. Necrotic tendon lying within granulation tissue containing small discrete deposits of lime.

Within this zone and filling the cavity of the crescent is a mass of tissue appreciably more cellular. The general appearance is that of an early form of granulation tissue. The predominant cell type is the round or plasma cell. There are a small number of spindle shaped connective-tissue cells and a few old and many newly formed bloodvessels. This tissue is abundantly infiltrated with red blood cells, most of which show degeneration changes, showing that the hemorrhage antedates the operation. The few old bloodvessels are small in size, and have thickened walls; this thickening is largely due to swelling of the intima. The newly formed bloodvessels are irregular in shape and size, and consist mainly of clefts or spaces within the granulation tissue lined by a distinct and uniform row of endothelium.

Upon the innermost aspect of this area of young granulation tissue there are finely granular amorphous masses of necrotic tissue. While this necrotic tissue stains diffusely with eosin, here and there are scattered discrete sand-like particles, that stain blue with hematoxylon, indicating a lime reaction. The greater part of this necrotic tissue lies loosely within the cavity of the crescent; a much smaller portion is still in direct continuity with the adjacent granulation tissue.

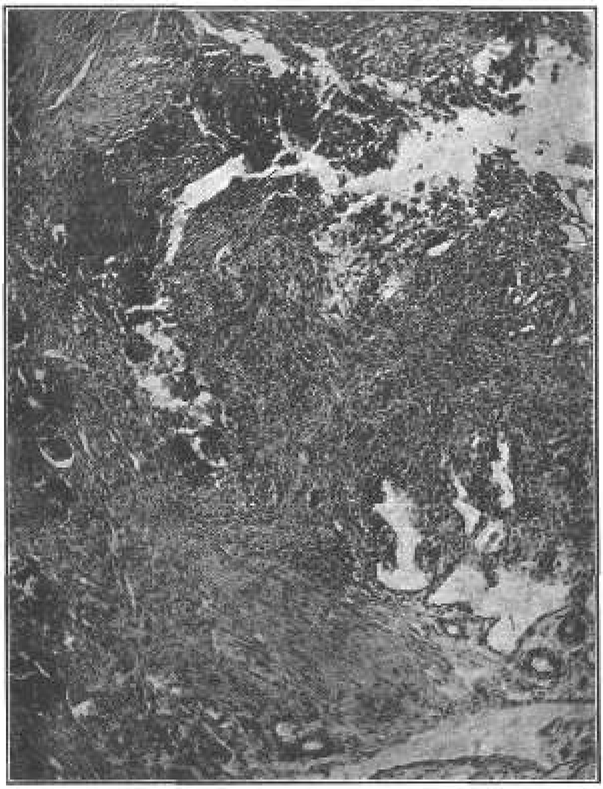

FIG. 3.—Case 2. Well-organized granulation tissue springing from a tendon that has undergone extensive hyaline degeneration. Small discrete lime deposits lying within the tendon and in the granulation tissue. Here and there a foreign body giant cell.

Epicrisis. This section reveals substantially two stages of granulation tissue: an outer, the older, which may be interpreted as representing the wall of a focus of inflammation; and an inner, a more recent variety. In addition, there is within the centre of this focus necrotic tissue. This has undergone partial calcification.

Diagnosis. Necrosis of tendon with partial calcification and young granulation tissue.

CASE II.—Indirect trauma four or five months previously. Male, aged thirty years. Extensive adhesions in bursa; fingernail-sized, gritty, yellow deposit entirely within supraspinatus tendon about 5 cm. from its insertion.

Pathological Examination. Sections show two different masses (Figs. 3 and 4). The first and larger consists of an irregular mass of tendon tissue continuous with a large area of granulation tissue. The tendon consists of firm fibrous longitudinal bands of connective-tissue, portions of which show hyaline degeneration. Only a small area of tendon fiber is present; the rest have undergone extensive calcification. The calcification is present either in the form of discrete, sharply circumscribed, sand-like masses embedded, as it were, in a cavity within the tendon; or as diffuse finely granular masses, shading imperceptibly into the surrounding tissue structure; or as finely granular amorphous material lying within the tendon or the granulation tissue.

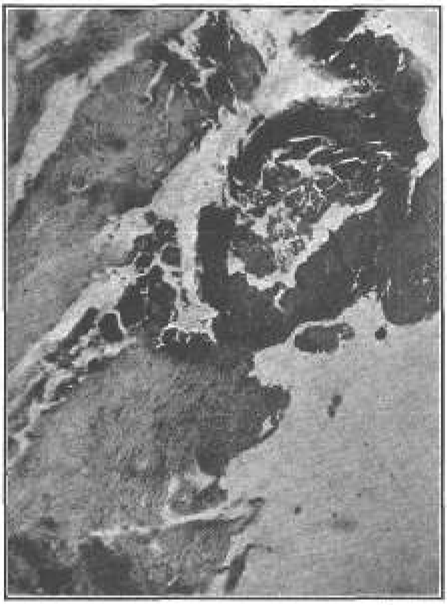

FIG. 4.—Case 2. Tendon tissue that has undergone extensive hyaline degeneration and calcification.

The granulation tissue is richly cellular, almost sarcomatous in appearance, and consists of large oval or spindle-shaped cells with a finely fibrillar intercellular connective-tissue. There are many delicately walled newly formed bloodvessels. A few giant cells are present, some in relation to the lime granules, some not. They are all probably so-called "foreign body" giant cells.

The second and smaller mass consists of hyaline tendinous tissue which has undergone almost complete calcification. The lime deposit consists of finely granular masses, either within the tendon or upon its surface.

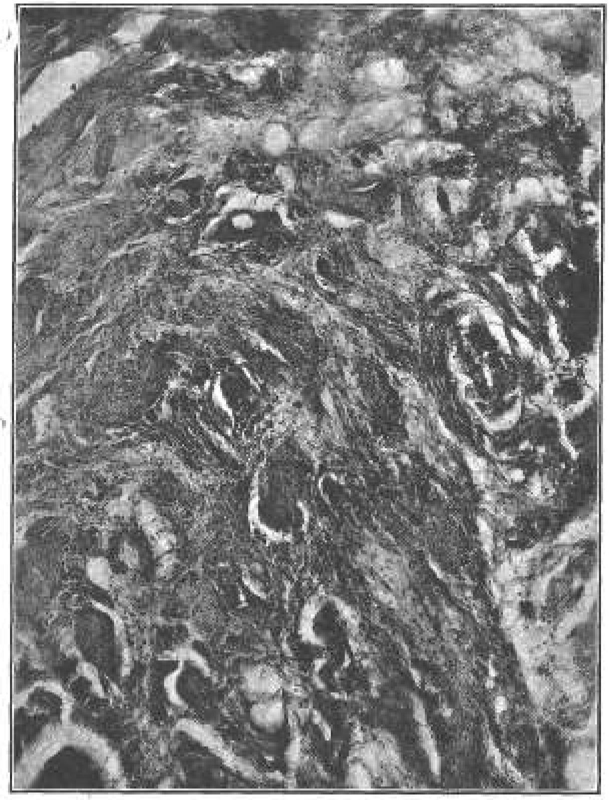

FIG. 5.—Case 3. Tendon bundles separated from one another by extensive scar tissue.

Epicrisis. A tendon that has undergone extensive hyaline degeneration and calcification. There is manifest necrosis with secondary calcification of the tendon. In addition, there is a comparatively early form of granulation tissue.

Diagnosis. Chronic tendinitis, with necrosis, hyaline degeneration, and calcification, and the formation of granulation tissue.

CASE III.—Chronic (one and a half years). Female, aged about forty-five years. No history of trauma. Persistent "brachial neuritis." A few fine adhesions in bursa; small, dry, gritty, yellow-white lime deposit, entirely within infraspinatus tendon near its insertion.

Pathological Examination. With the low objective the section shows essentially a mass of tendon tissue encircling a cavity which contains necrotic detritus (Figs. 5 and 6). The tendon tissue consists of dense strands of fibrous tissue which is grouped in longitudinal strands or bundles. Between many of these bundles is loose cellular tissue, consisting of coarse fibrous tissue, many spindle-shaped connective-tissue cells, and a small number of delicate-walled bloodvessels. In some areas the tendon bundles have undergone hyaline degeneration. The cavity within the tendon contains a finely granular amorphous mass of necrosed material which in part lies loosely within the cavity and in part is continuous with the fibrous wall of the cavity. No calcification is present, although Dr. Brickner assures me that much gritty material was found at operation. The lime must have been shaken out of the specimen during manipulation.

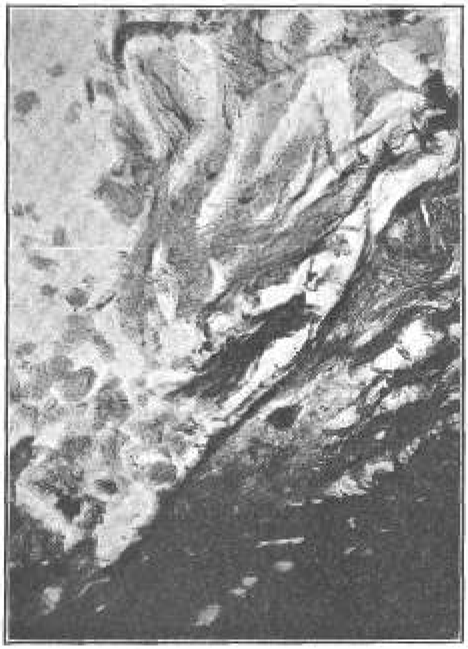

FIG. 6.—Case 3. Wall of cavity within tendon containing necrotic tendon tissue.

Epicrisis. This section represents a tendon between the bundles of which firmly organized tissue has formed. The tendon bundles have in part undergone hyaline degeneration. Within the tendon is a cavity containing necrosed material.

Diagnosis. Chronic tendinitis with hyaline degeneration.

CASE IV.—Acute. Male, aged about sixty years. Operation seventeen days after direct injury. Well-developed adhesions in bursa; two discrete white powdery deposits in supraspinatus tendon.

Pathological Examination. Sections reveal a thin rind of tendon tissue enclosing a cavity containing finely granular necrosed material. Within the tendon tissue are extensive areas of necrosis, most of which take the lime reactions with hematoxylon. The necrotic material within the cavity also shows extensive areas of calcification. In no specimen was the slightest reactive tissue noted, in the form of round-celled infiltration, granulation tissue, or the formation of new bloodvessels.

Diagnosis. Necrosis and calcification of tendon.

SUMMARY. The first three cases exemplify three stages of a lesion that may be termed "tendinitis." The common factor is the formation within and around the tendon fibers of granulation tissue which corresponds in a general way, according to morphological criteria, with the duration of the illness. We find, accordingly, a progressive organization of the reactive inflammatory tissue in and around the necrosed portions of the tendon, so that a mere round-celled infiltration with early formation of bloodvessels in the first case becomes a scar-like tissue, with fully developed bloodvessels in the third. The last case shows no reactive tissue around the dead portions of the tendon, although the duration of illness was practically the same as in the first case. The only way we can account for this absence is the age of the patient, at which time the reaction forces of the body are notoriously deficient.

Necrosis. Another common factor in all four cases is the presence of necrotic tissue. It has the same morphological appearance in all, namely, a finely granular, amorphous, and completely acellular mass. In Cases I, II, and IV this necrotic tissue is more or less calcified. Topographically the necrotic tissue has two relations: either in direct continuity with the tendon, or with a greater or smaller zone of granulation tissue intervening.

The necrotic tissue can have only two sources: (1) the degeneration of the blood and serum consequent upon the primary trauma; (2) the breaking down by coagulation necrosis of the tendinous structures. The first origin can obviously be excluded for the following reasons: (*a*) There is no blood pigment; (*b*) the appearance of the tissue is too homogeneous and plastic to arise from a fluid origin; (*c*) the continuity between the necrosed area and the surrounding solid tissue is a gradual one, so that it is impossible to determine a sharp definition between the well-preserved and the necrotic areas. Were the necrosis of fluid origin we should expect a cyst-like cavity. I conclude, therefore, that the necrosis arises from actual death of tendon. The explanation of the necrosis is obvious. The blood supply of a tendon is notoriously meager. The tendon bundles proper have no blood supply. Whatever bloodvessels the tendon contains are present only in the connective-tissue left between the tendon bundles (Stöhr), and these are small in size and number. It is, therefore, readily understandable how a trauma, however slight, may interfere profoundly with the blood supply of a given area of tendon and thereby cause necrosis.

Calcification. Lime is found in Cases I, II, and IV. In Case III Dr. Brickner assures me that he found gritty masses at the operation. The failure to find lime microscopically, therefore, may be regarded as accidental, due, in all probability, to the fact that the lime deposit was shaken out in the subsequent manipulation of the specimen.

The lime is present in three forms: (1) as small sand-like particles within the necrosed tissue; (2) as diffuse massive calcification of the necrosed tissue; (3) as isolated discrete and sharply defined nodules embedded within the tendon or the granulation tissue. Furthermore, Brickner reports calcification in some cases upon the surface of the tendon, in the form of a thick, white, gritty material, associated with a minute tear of the tendon. He has also found at operation, and by Roentgen-ray, multiple deposits in the tendon, sometimes at a considerable distance from its insertion. In some cases the shadow-forming deposit is quite liquid, and this occurs in early as well as old cases, in extra- as well as intratendinous deposits.

What is the pathogenesis of this calcification? At the outset it is necessary to settle the question whether these tendinous calcifications are due to injury of the humerus, by the statement that neither Brickner nor any other observer has ever seen any direct connection of the deposit with the bone, either by Roentgen rays or at operation.

The pathogenesis of calcification within tendons is governed by the same conditions that apply to abnormal calcifications in other parts of the body. It has been definitely established that calcification only occurs in dead or inert tissue, that is, tissues that have no blood supply. It never occurs in tissues whose blood supply is abundant. Familiar examples of calcification in necrosed tissues are those in caseous tubercles, atheromatous plaques, old abscesses, infarcts, and old sebaceous and dermoid cysts. Furthermore, calcification has a peculiar predilection for connective-tissues that have undergone hyaline degeneration, which also is merely a form of necrosis. This is commonly noted in fibromyomata of the uterus of long standing. I have also noted this phenomenon in the hyaline corpora albicantia of the ovary; in the wall of a chronically inflamed tube; in old pleuritic thickenings; in the walls of hydatid cysts; in old scars, etc. Furthermore, it is an interesting observation that wherever we find a deposit of lime, we are apt, at the same time, to find partial ossification of the tissues. The conditions under which ossifications form after calcification are not precisely understood, but they probably depend upon two factors: (1) a sufficient period of time and (2) the formation of a reactive blood supply. We predict, therefore, that an extensive study of calcified tendons will sooner or later discover an instance of ossification. This point will again be discussed in greater detail.

Causation of Early Calcification. Brickner[2] calls attention to the remarkably early appearance of the calcification in these cases. In addition to the first and fourth cases, in which the lime deposit was demonstrated, roentgenographically, upon the tenth and eleventh days respectively following the injury, Brickner has found the deposit by roentgenography as early as twenty-four hours and five days after trauma.

Now, in all the instances of pathological calcification that we have considered it has been generally accepted that the deposition of lime occurs only when the necrosis is of long standing. Calcification of a tissue, in other words, is generally regarded as indicating chronicity of the lesion. How, therefore, are we to account for the very early calcification noted in these cases?

It seems to me that sufficient explanation of this phenomenon is found in the observations upon experimental calcification. Litten[3] and Lick[4] found lime microscopically in necrosed portions of the kidney, one day after ligature of the renal artery. Especially significant for our purposes are the observations of Schujeninoff,[5] who *found calcification of the muscle fibers both in man and in* animals one and a half days after the suture of a divided muscle. The lime in every instance was near the suture line, and the affected muscle fiber was the seat of colloid degeneration. Schujeninoff believed that the calcification was due to interference with the circulation by the ligation of small bloodvessels. I have frequently seen calcification of necrosed portions of malignant growths when the duration of necrosis could be reasonably reckoned in terms of days.

Strangely enough, the repeated observations upon experimental healing of tendons fail to throw any light upon the problem of the early incidence of calcification in the spinatus tendons. Studies upon the injury and repair of tendons are numerous enough, the more comprehensive being those of Palla,[6] Seggel,[7] and Yamagiva.[8] I have studied the extensive protocols of these writers carefully, and in not a single instance was calcification of the tendon noted. The absence of calcification is puzzling, and can be accounted for only by the fact that the tendo Achilles, exclusively employed for experimental purposes, presents some peculiar biological properties that do not predispose it to calcification. In order to satisfy ourselves upon this point, we propose, at some future date, to repeat these experiments upon the spinatus tendons.

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[2] Loc. cit.

[3] Ztschr. f. klin. Med., 1880, v. i.

[4] Arch. f. klin. Chir., 1908, lxxxv, 118.

[5] Ztschr. f. Heilk., 1897, xviii, 79.

[6] Beitr. z. klin. Chir., 1909, lxiii, 644.

[7] Ibid., Chir., 1903, xxxviii, 342.

[8] Arch. f. Path. Anat., Virchow, cxxxv, 308.

At all events, there is already ample evidence in certain tissues, at least, that calcification in necrotic tissue occurs earlier than is generally believed. In all probability the early or late incidence of calcification depends upon peculiar predilections of certain tissues, due to chemical affinities the nature of which has not been precisely determined (Adami).[9] This will explain why calcification occurs oftener in some tissues than in others. Wells[10] accounts for the greater frequency (in his experience) of calcification in the lungs, stomach, and kidneys by the fact that these organs have an acid secretion which precipitates carbonate of lime. This contention is hardly tenable in view of the fact that some of the commonest lime deposits arise in tissues that are constantly bathed in alkaline juices, for example, arteriosclerosis. To explain the early calcification in tendons, we suggest that perhaps it is the collagen, present in the tendons, according to Gies and Buerger,[11] to the extent of 37 per cent., that acts as the lime precipitant. This suggestion arose from the observations of Schujeninoff,[12] who showed that the muscle fibers in which lime deposition occurred were in a state of colloid degeneration—a condition chemically allied to collagen; and, furthermore, by the affinity of cartilage (which is also rich in collagen, (Hammersten[13]) for calcification.

Maturity of the patient is unquestionably a large factor in determining calcification of tendon, and, indeed, of other tissues as well. Calcification is extremely rare in children and youth, and becomes progressively more frequent as age advances. This explains why subacromial bursitis with calcification of the spinatus tendons has never been observed in the young.

Previous Pathological Studies upon Calcification of the Spinatus Tendon. As far as we are aware, no previous systematic study has been made of the pathogenesis of the lesion underlying the symptom-complex embraced under the term "subacromial bursitis." There are only two pathological reports upon excised portions of tendons, that of Wright, in one of Codman's[14] cases and that of Wrede.[15]

Wright's report was "microscopic examination of the material from your case of 'sub-deltoid bursitis' shows the following: The main constituent of the material is a coarse, dense, fibrous tissue. Associated with this is a looser connective-tissue in which are enclosed bloodvessels and a good many cells of the lymphocyte series. The coarse, fibrous tissue in some places contains more fibroblasts than elsewhere, and may therefore be considered to be in process of growth at these points. In a few places it is necrotic, and it contains few bloodvessels. I can not find any evidence of tuberculosis. The material seems to me to show a slow, productive inflammatory process combined with degeneration and necrosis similar to the process observed in the heart valves in some cases of chronic endocarditis."

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[9] Principles of Pathology, Philadelphia, 1914.

[10] Arch. Int. Med., vii, 721.

[11] Amer. Jour. Physiol., December, 1901.

[12] Loc. cit.

[13] Text-book of Physiological Chemistry, Trans. from 7th German ed., 1911.

[14] Boston Med. and Surg. Jour., 1908, clix, beginning October 22.

[15] Arch. f. klin. Chir., 1912, xcix, 259.

Wrede's case: Man, aged seventy-six years, who had suffered for one year following a trauma. At operation a deposit within the supraspinatus tendon was found. From this portion a small piece was excised. The tendon was found necrotic with deposition of lime. In addition there were granulation tissue and giant cells. These findings, therefore, correspond to our own. Wrede enters into a prolix discussion of the etiology of the malady, and finally assumes a non-committal attitude.

Analogy of the Lesion to Other Pathological States of Tendons. Calcification of the spinatus tendons, following so soon after trauma (the etiology described by the more extensive observers of this malady, Brickner and Codman), has, as far as I am aware, no precise analogy in human pathology. This singularity is especially striking when we consider that the trauma in most of the cases is slight or trivial, merely sufficient, as Brickner and Codman declare, to jam the spinatus tendons between the head of the humerus and the acromion process. The thought obviously suggests itself, Why are other tendons, such as the tendo Achilles and those around the knee and elbow which are more frequently subjected to trauma, free from such calcification? The answer is that such calcifications probably do occur, although the pathological proof is thus far lacking.

Since the introduction of the Roentgen rays, shadows in tendons, especially in the neighborhood of joints, are a frequent finding. As instances, we cite the not uncommon calcification or ossification in the tendo Achilles arising in continuity from the posterior aspect of the os calcis (Höring,[16] Jacobsthal,[17] Haglund,[18]), but sometimes isolated within the tendon (Blencke,[19] Finney,[20]); also to the frequent ossification of tendons around joints following sprain or fracture, for example, the triceps (Engelbrechtson,[21] Fragenheim[22]), brachialis anticus (Tyson[23]), adductor longus (Boyd,[24] Whitelock[25]), obliquus externus (Charles[26]), quadriceps femoris (Bähr[27]), gastrocnemius and deltoideus (Whitelock[28]). The cases of so-called "riders' bone," *i. e.*, formation of bone within the tendons of the adductors of the thigh, and "drill bone," in the insertion of the deltoid, probably also belong to this group of traumatic "ossifications" of tendons. These citations prove that traumatic "ossifications" do occur, although by no means commonly. I have purposely cast doubt upon the validity of the osseous formation by the use of quotation marks for the reason that, with the exception of the case of ossification of the tendo Achilles, reported by Jacobsthal, the pathological proof of the presence of bone is entirely lacking. It is by no means proved that some of the shadows reported as bone by various observers may not have been those of lime concretions.

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[16] Deutsch. med. Wchnschr., 1908, xxxiv, 175.

[17] Arch. f. klin. Chir., lxxxviii, 146.

[18] Ztschr. f. orthop. Chir., xix, 457.

[19] Ibid, 1908, xx, 363.

[20] Quoted by Binnie, Keen's Surgery, vol. ii.

[21] Railway Surg. Jour., 1911–1912, xviii, 320.

[22] Med. Klinik, 1909, v, 278.

[23] British Med. Jour., April 16, 1910.

[24] Clin. Jour., London, 1897, x, 186.

[25] Sprains and Allied Injuries of Joints, London, 1909.

[26] British Med. Jour., 1912, i, 949.

[27] Quoted by Engelbrechtson.

Whether these shadows are of lime or bone is, for our purposes, a matter of indifference, for the reason that, as most pathologists have agreed, calcification always precedes abnormal ossification. These shadows at some stage of the process must have been those of pure lime deposits. The earlier stages of bone formation are precisely those that we have described in our tendon specimens, namely, death of tissue with calcification. I have never, nor, as far as I am aware, has anybody else, seen an area of ossification occurring *de novo* in normal tissue.[29]

This postulate leads us to predict, as I have said before, that when more of these calcified spinatus tendons are examined, bone will sooner or later be found.

The demonstration, therefore, that calcification always precedes ossification removes whatever doubt may have arisen as to the validity of the analogy between the traumatic calcification of the supraspinatus tendon and the supposed bony lesions that we have enumerated.

The mere demonstration of an analogous traumatic lesion of the tendons by no means accounts, however, either for the peculiarly common or for the early incidence of calcification of the spinatus tendons. The lesions in the other tendons to which I have referred are infrequent, and the manner of the process indicates that the calcification must have come rather late upon the trauma. The lesions in the spinatus tendons, on the other hand, are comparatively common, as proved by the experience of Brickner, who has seen over twenty cases within the last two years. The early and common incidence appearance of lime in these cases has no precise counterpart, and neither of us is able to submit a satisfactory explanation. We hope to be able to later.

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[28] Loc. cit.

[29] I have summarized the entire subject of the histogenesis of ossification in a paper shortly to be published, entitled, "The Relation of Angiogenesis to Ossification." In passing, the manner whereby a mass of lime is converted into bone is an interesting process, and analogous in its details to normal endochondral or membranous ossification. Furthermore, I shall show that the development of bloodvessels forms the keynote to the interpretation of the entire process.